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INSTITUTE REPORT NO. 239

WHAT'S WRONG WITH THE WOUND BALLISTICS LITERATURE, AND WHY

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DIVISION OF MILITARY TRAUMA RESEARCH

JULY 1987

LETTERMAN ARMY INSTITUTE OF RESEARCH
PRESIDIO OF SAN FRANCISCO, CALIFORNIA 94129

UNCLASSIFIED
SECURITY CLASSIFICATION OF THIS PAGE

REPORT DOCUMENTATION PAGE

Form Approved
OMB No 0704-0188
Exp. Date Jun 30, 1986

1a. REPORT SECURITY CLASSIFICATION UNCLASSIFIED	1b. RESTRICTIVE MARKINGS
2a. SECURITY CLASSIFICATION AUTHORITY	3. DISTRIBUTION/AVAILABILITY OF REPORT This document has been approved for public release and sale. Unlimited.
2b. DECLASSIFICATION/DOWNGRADING SCHEDULE	
4. PERFORMING ORGANIZATION REPORT NUMBER(S)	5. MONITORING ORGANIZATION REPORT NUMBER(S)
6a. NAME OF PERFORMING ORGANIZATION Letterman Army Institute of Research 6b. OFFICE SYMBOL (If applicable) SGRD-UL-MT	7a. NAME OF MONITORING ORGANIZATION US Army Medical Research and Development Command
6c. ADDRESS (City, State, and ZIP Code) Division of Military Trauma Research, LAIR Presidio of San Francisco, CA 94129-6800	7b. ADDRESS (City, State, and ZIP Code) Fort Detrick, Frederick, MD 21701
8a. NAME OF FUNDING/SPONSORING ORGANIZATION 8b. OFFICE SYMBOL (If applicable)	9. PROCUREMENT INSTRUMENT IDENTIFICATION NUMBER
8c. ADDRESS (City, State, and ZIP Code)	10. SOURCE OF FUNDING NUMBERS PROGRAM ELEMENT NO.: 3S16277 PROJECT NO.: 2A874 TASK NO: AC WORK UNIT ACCESSION NO: 109

11. TITLE (Include Security Classification)
Institute Report No. 239, What's Wrong with the Wound Ballistics Literature, and Why

12. PERSONAL AUTHOR(S)
M.L. Fackler, M.D.

13a. TYPE OF REPORT	13b. TIME COVERED	14. DATE OF REPORT (Year, Month, Day)	15. PAGE COUNT
Institute Report	FROM 1986 TO 1987	1987 July	33

16. SUPPLEMENTARY NOTATION

Keywords:

17. COSATI CODES			18. SUBJECT TERMS (Continue on reverse if necessary and identify by block number)
FIELD	GROUP	SUB-GROUP	Wound Ballistics; Gunshot Wounds; High Velocity; Kinetic Energy

19. ABSTRACT (Continue on reverse if necessary and identify by block number)

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20. DISTRIBUTION/AVAILABILITY OF ABSTRACT	21. ABSTRACT SECURITY CLASSIFICATION	
☒ UNCLASSIFIED/UNLIMITED ☐ SAME AS RPT. ☐ DTIC USERS	UNCLASSIFIED	
22a. NAME OF RESPONSIBLE INDIVIDUAL Martin L. Fackler	22b TELEPHONE (Include Area Code) (415) 561-5817	22c OFFICE SYMBOL SGRD-UL-MT

DD FORM 1473, 84 MAR 83 APR edition may be used until exhausted. All other editions are obsolete

SECURITY CLASSIFICATION OF THIS PAGE
UNCLASSIFIED

ABSTRACT

Attempts to explain wound ballistics (the study of effects on the body produced by penetrating projectiles) have succeeded in mystifying it. Fallacious research by those with little grasp of the fundamentals has been perpetuated by editors, reviewers, and other investigators with no better grasp of the subject. This report explains the projectile-tissue interaction and presents data showing the location of tissue disrupted by various projectiles. These tissue disruption data are presented in the form of wound profiles. The major misconceptions perpetuated in the field are listed, analyzed, and their errors exposed using wound profiles and other known data. The more serious consequences of these misconceptions are discussed. Failure in adhering to the basic precepts of scientific method is the common denominator in all of the listed misconceptions.

Gunshot wounds are a fact of life in our society. The common assumption is that military conflicts, wound ballistics research, and a steady stream of daily experience in our larger cities have provided the knowledge and skill to assure uniform excellence in treatment of these injuries. Sadly, this assumption is wrong.

Probably no scientific field contains more misinformation than wound ballistics. In a 1980 Journal of Trauma editorial entitled "The Idolatry of Velocity, or Lies, Damn Lies, and Ballistics," Lindsey identified many of the misconceptions and half-truths distorting the literature (1). Despite his cogent revelations, the errors he attempted to rectify are still being repeated in the literature (2-7), often embellished with unproven assumption and uninformed speculation. The body of literature generated at the wound ballistics laboratory of the Letterman Army Institute of Research over the past six years (8-14) strongly supports the points made by Professor Lindsey. The author of this paper has chosen to correct errors, as they appeared, with letters to journal editors (15-22), a time-consuming endeavor of questionable effectiveness. This critical review calls attention to the problem, corrects the most widespread and damaging misinterpretations, and lays the groundwork for improved research, understanding and clinical treatment.

Between 1875 and 1900, the study of gunshot effects had reached a high level of sophistication, thanks mainly to Theodor Kocher, whose work was the epitome of sound scientific method (23-27). However, with the advent of the high-speed movie camera in the present century, emphasis in wound ballistics shifted from sound scientific method to spectacular cinematography--a triumph of high technology over common sense. Unfortunately, a sideshow mentality seized upon the technology of the twentieth century. Flamboyance attracted more attention than sound science. Wound ballistics research was reduced to taking movies of shots into everything imaginable, and the focus of understanding narrowed to exclude every variable except projectile velocity. The exaggeration inherent in these methods so distorted the concept of temporary cavitation that, to some, it came to represent the entirety of the projectile-tissue interaction (28, 29). Rarely does the viewer find a measuring scale included in reproductions of these dramatic cinematographic frames (30). Undoubtedly, many readers have seen the Swedish film of an anesthetized

pig being shot through the abdomen with an M-16 rifle that "made the rounds" about fifteen years ago. No scale or any other item was included to provide size orientation. How large was the pig? Most would assume the animal to be in the 100- to 150-kg range. It was actually a mini-pig, weighing about one tenth that much. The exaggeration of effects so introduced is obvious.

THE WOUND PROFILE - UNDERSTANDING THE PROJECTILE-TISSUE INTERACTION

A projectile crushes the tissue it strikes during penetration, and it may impel the surrounding tissue outward (centrifugally) away from the missile path. Tissue crush is responsible for what is commonly called the permanent cavity and tissue stretch is responsible for the so-called temporary cavity. These are the sole wounding mechanisms. In addition, a sonic pressure wave is generated by projectiles traveling faster than the speed of sound. In air this wave trails the projectile like the wake of a ship. The sonic boom experienced after passage of a supersonic airplane is an example of a sonic pressure wave. This pressure wave travels at the speed of sound in the medium through which it passes, and sound travels four times as fast through tissue as it does through air. Thus the sonic wave precedes the projectile in tissue. Contrary to popular opinion (3, 30), this wave does not move or injure tissue. Harvey's exhaustive experiments during WW II showed clearly the benignity of the sonic pressure wave (31). The lithotripter, a recent invention that uses this sonic pressure wave to break up kidney stones, generates a wave five times the amplitude of the one from a penetrating small arms projectile. Up to 2,000 of these waves are used in a single treatment session, with no damage to soft tissue surrounding the stone (32,33). It would be difficult to imagine more convincing confirmation of Harvey's conclusions.

The wound produced by a particular penetrating projectile is characterized by the amount and location of tissue crush and stretch. In our laboratory, we measure the amount and location of crush (permanent cavity) and stretch (temporary cavity) on the basis of shots fired into gelatin tissue simulant. Since we have calibrated this simulant to reproduce the projectile characteristics (penetration depth, deformation, fragmentation, yaw) equivalent to those observed in living animal tissue,

measurements from these shots can be used to predict approximate animal tissue disruption (8-10). These data are presented in the form of Wound Profiles (Fig 1-8), which illustrate the amount, type, and location of tissue disruption, projectile mass, velocity, construction, and shape (before and after the shot), as well as projectile deformation and projectile fragmentation pattern when applicable. The scale on each profile permits quick determination of tissue disruption dimension at any point along the penetration path for comparison with other profiles, other experimental results, or with measurements from actual wounds in a clinical setting or at autopsy. Wound profile data will be used to rectify the fallacies listed below.

MAJOR MISCONCEPTIONS

1. Idolatry of Velocity:

A widespread dogma claims that wounds caused by "high-velocity" projectiles must be treated by extensive excision of tissue around the missile path (34-40), whereas those caused by "low-velocity" missiles need little or no treatment (41, 42). Two half-truths nurture this error. The first of these, "Cavitation is a ballistic phenomenon associated with very high velocity missiles" (7), is easily disproved. The wound profile in Fig 1 shows a very substantial temporary cavity produced by a "low-velocity" bullet. This bullet, fired from the Vetterli rifle at 1357 ft/s (414 m/s), has ballistic characteristics typical of those used by military forces in the latter half of the nineteenth century. It is the same bullet used by Theodor Kocher for most of his wound ballistics studies (23-27). It is obvious from this wound profile that temporary cavitation is not, as popularly believed, a modern phenomenon associated exclusively with projectiles of "high velocity."

The adjunct half-truth, "Cavitation requires extensive debridement of tissues..." (7), lacks valid scientific support. Cavitation is nothing more than a transient displacement of tissue, a stretch, a localized "blunt trauma." It is not surprising that elastic tissues such as bowel wall, lung, and muscle are relatively resistant to being damaged by this stretch, while solid organs such as liver are not (9). Most of the muscle subjected to temporary cavity stretch survives; tissue survival has been verified in every case in which muscle

was allowed to remain in situ and healing was followed to completion (43-48).

Misinterpretation of the mechanism by which the M-16 rifle causes tissue disruption perpetuated the foregoing misconceptions. The M-16 (Fig 2) was introduced in Vietnam, and many compared the increased tissue disruption it produced (12-14, 49, 50) with that caused by previous military rifles. In the Vietnam era, the major role played by bullet fragmentation in tissue disruption was not recognized (8). It is now appreciated (12-14) and documented (Fig 3) that bullet fragmentation is the predominant reason underlying the M-16's increased tissue disruption. Despite this recent evidence, a generation of surgeons and weapon developers (28) has been confused and prejudiced by the assumption that "high velocity" and "temporary cavitation" were the sole causes of tissue disruption.

It is indeed surprising that only Lindsey questioned the attribution of the marked increase in tissue disruption to a rather modest 10% increase in velocity. Surely, someone should have noticed that the largest increase of projectile velocity in the history of small-arms development (a 50% increase--made possible by the invention of the copper-jacketed bullet near the end of the nineteenth century) was accompanied by a marked decrease in soft tissue disruption (51, 52). This decrease was predicted by Kocher, whose work had taught him the importance of projectile deformation (26, 27); new smaller-caliber bullets did not deform upon striking tissue as did previous large caliber soft lead bullets (Fig 1).

2. Exaggeration of Temporary Cavity Size, Pressure, and Effect:

In 1971, Amato et al (53) wrote that the temporary cavity "can approximate 30 times the size of the missile." They showed the temporary cavity caused by a 0.25-in. (6.4-mm) steel sphere shot at 3,000 ft/s (914 m/s) through the hind leg of an anesthetized dog. Although no scale was included on the high-speed roentgenograms, the reader can use dividers to determine the sphere diameter and will find that the largest temporary cavity shown is 11 sphere diameters--not 30 diameters. Wound profile data obtained in our laboratory gave comparable results; a 6-mm steel sphere at slightly over 1000 m/s produced a maximum

temporary cavity of only 12.5 sphere diameters (Fig 4). Other authors, citing no data, describe the temporary cavity as "...30 times the diameter of the projectile..." (35), "...30 times or more..."(54), and 30 to 40 times the missile diameter (36, 40)--all sizable exaggerations.

To further confuse the issue, pressures of up to 100 atmospheres are incorrectly attributed to temporary cavitation by many authors (39, 40, 55-57). These authors appear to have confused the sonic pressure wave with the pressure generated in tissues by temporary cavitation. Temporary cavity tissue displacement can cause pressures of only about 4 atmospheres (31). A careful reading of Harvey's paper (31) should correct this confusion.

Probably the most exaggerated account of temporary cavity effect in the literature appears in High Velocity Missile Wounds by Owen-Smith (36). His Fig 2.20 on page 35 shows a lesion in a pig's colon caused by a "standard bullet fired at 770 m/s (2500 ft/s)." Concerning this wound, he states "there are microscopic changes of cell death extending 20 cm from the edge of the hole in the colon; this is why such an area must be resected if it has been damaged by a rifle bullet." Perusal of the source document of this picture (58), however, reveals that a deforming soft-point hunting bullet was used for this shot. In describing the effect of this shot, the source document states, "...haemorrhage extended macroscopically to a diameter (my emphasis) of 20 cm." When the 8-cm hole diameter is subtracted, a 6-cm distance (rather than the 20 cm reported by Owen-Smith) from the edge of the hole on each side adds up to the "diameter of 20 cm" reported by Scott in the source document. Furthermore, photographs of bowel defects caused by bullets must be viewed with caution. Folding back the bowel wall around the edges of the hole can make tissue defects appear larger. If colon tissue at a distance of 20 cm from the bullet hole is killed, as asserted by Owen-Smith, what happens to the loops of small bowel and other organs that are within 20 cm of the bullet hole? Are they killed too? If so, this would equate to destruction of most of the abdominal contents by every penetrating "high-velocity" bullet. Clearly, this conclusion is inconsistent with well established available facts. A study done in our laboratory (9), for example, showed damage to a pig colon caused by a nondeforming military bullet traveling at 911 m/s (2989 ft/s) that was only slightly larger than the dimensions of the bullet that had caused it.

It should be noted, however, that stretch from temporary cavity tissue displacement can disrupt blood vessels or break bones at some distance from the projectile path (40), just as they can be disrupted by blunt trauma. We can produce this in the laboratory by careful choice of projectile and projectile trajectory in tissue (48), but in practice this happens only very rarely. Data from the Vietnam conflict show that the great majority of torso and extremity wounds were attributable to the damage due to the permanent cavity alone (59).

3. Assumption of Bullet "Tumbling" in Flight:

The notion that a common cause of increased wounding is the bullet's striking at large yaw angles (angle between the bullet's long axis and line of flight), or even sideways due to "tumbling" in flight (37, 40), is clearly fallacious. Anyone who has ever shot a rifle and observed the holes made by the bullet recognizes that they are round, not oblong, as would be the case if they yawed or tumbled in flight. This misconception seems attributable in large measure to misinterpretation of a report published, in 1967, by Hopkinson and Marshall. These authors presented diagrams of the yaw angles and patterns made by the bullet tip in flight (60). The angles on their drawings were exaggerated for clarity, showing 25 to 30 degrees rather than the 1 to 3 degrees that actually occur for properly designed bullets of small arms (61). In 1972, Amato and Rich reproduced these diagrams and added one for "tumbling" (62). In 1975 these diagrams reappeared in the NATO Handbook-Emergency War Surgery (40), where the text described them as resulting from aerodynamic forces acting upon the spin-stabilized bullet during flight. In 1980, Swan and Swan (37) reproduced these diagrams, but for the yawing bullet showed the impossible situation of rotation around the bullet tip rather than its center of mass. They also added a unique opinion (unsupported) that "yaw" and "tumble" are special ballistic properties associated with missiles of "very high velocities (c [sic] 3000 ft/s)."

Data from ballistics studies (10, 13, 14) show quite clearly that:

- Bullets fired from a properly designed rifle yaw no more than a few degrees in flight, regardless of velocity.

- In their path through tissue, all nondeforming pointed bullets, and some round-nosed ones, yaw to 180 degrees, ending their path traveling base forward (Figs 3 and 5).

Thus bullet yaw in tissue, an important consideration, has been confused with bullet yaw in flight, which is, in most cases, of negligible consequence.

4. Presumption of "Kinetic Energy Deposit" to Be a Mechanism of Wounding:

Serious misunderstanding has been generated by looking upon "kinetic energy transfer" from projectile to tissue as a mechanism of injury. In spite of data to the contrary (1, 63), many assume that the amount of "kinetic energy deposit" in the body by a projectile is a measure of damage (2-5, 36, 37, 40). Such opinions ignore the direct interaction of projectile and tissue that is the crux of wound ballistics. Wounds that result in a given amount of "kinetic energy deposit" may differ widely. The nondeforming rifle bullet of the AK-74 (Fig 6) causes a large temporary cavity which can cause marked disruption in some tissue (liver), but has far less effect in others (muscle, lung, bowel wall) (9). A similar temporary cavity such as that produced by the M-16 (Fig 2), stretching tissue that has been riddled by bullet fragments, causes a much larger permanent cavity by detaching tissue segments between the fragment paths. Thus projectile fragmentation can turn the energy used in temporary cavitation into a truly destructive force because it is focused on areas weakened by fragment paths rather than being absorbed evenly by the tissue mass. The synergy between projectile fragmentation and cavitation can greatly increase the damage done by a given amount of kinetic energy.

A large slow projectile (Fig 7) will crush (permanent cavity) a large amount of tissue, whereas a small fast missile with the same kinetic energy (Fig 4) will stretch more tissue (temporary cavity) but crush little. If the tissue crushed by a projectile includes the wall of the aorta, far more damaging consequences are likely to result than if this same projectile "deposits" the same amount of energy beside this vessel.

Many body tissues (muscle, skin, bowel wall, lung) are soft and flexible--the physical characteristics of a good shock absorber. Drop a raw egg onto a cement floor from a height of 2 m; then drop a rubber ball of the same mass from the same height. The kinetic energy exchange in both dropped objects was the same at the moment of impact. Compare the difference in effect; the egg breaks while the ball rebounds undamaged. Most living animal soft tissue has a consistency much closer to that of the rubber ball than to that of the brittle egg shell. This simple experiment demonstrates the fallacy in the common assumption that all kinetic energy "deposited" in the body does damage.

The assumption that "kinetic energy deposit" is directly proportional to damage done to tissues also fails to recognize the components of the projectile-tissue collision that use energy but do not cause tissue disruption. They are 1) sonic pressure wave, 2) heating of the tissue, 3) heating of the projectile, 4) deformation of the projectile, and 5) motion imparted to the tissue (gelatin block displacement for example).

The popular format for determination of "kinetic energy deposit" uses a chronograph to determine striking velocity and another to determine exit velocity. A 15-cm-thick block of tissue simulant (gelatin or soap) is the target most often used. This method has one big factor in its favor; it is simple and easy to do. As for its validity, the interested reader is referred to wound profiles shown in Figs 1-7. Comparing only the first 15 cm of the missile path with the entire missile path as shown on the profiles shows the severe limitation of the 15-cm block format. The assumption by weapons developers that only the first 15 cm of the penetrating projectile's path through tissue is of clinical significance (64) may simplify their job, but fails to provide sufficient information for valid prediction of the projectile's wounding potential. The length of bullet trajectories through the human torso can be up to four times as long as those in these small blocks. Even if this method were scientifically valid, its use has been further flawed by nearly all investigators who have included the M-16 rifle bullet in those projectiles tested. This method assumes that the projectile's mass remains constant through both chronographs. The M-16 routinely loses one third of its mass in the form of fragments which may remain in the target (see Fig 2). The part of the bullet that passes

through the second chronograph screens weighs only about two-thirds as much as the intact bullet that passed through the first set of screens. No provision is made for catching and weighing the projectile to correct for bullet fragmentation when it occurs. The failure to correct for loss of bullet mass can cause large errors in "energy deposit" data (8).

Surgeons sometimes excise tissue from experimental missile wounds that is, in their judgment, nonviable and compare the weight of tissue excised with the "kinetic energy deposited" (65). A surgeon's judgment and his technique of tissue excision is very subjective, as shown by Berlin et al (66), who found in a comparison that "One surgeon excised less tissue at low energy transfers and rather more at high energy transfers than the other surgeon, although both surgeons used the same criteria when judging the tissues." None of these experiments included control animals to verify that tissue the surgeon had declared "nonviable" actually became necrotic if left in place. Interestingly, all studies in which animals were kept alive for objective observations of wound healing report <u>less lasting tissue damage than estimated from observation of the wound in the first few hours after it was inflicted</u> (43-47, 67, 68). In a study of over 4,000 wounded in WW II it was remarked, "It is surprising to see how much apparently nonvital tissue recovered" (69).

Anyone yet unconvinced of the fallacy in using kinetic energy alone to measure wounding capacity might wish to consider the example of a modern broadhead hunting arrow. It is used to kill all species of big game, yet its striking energy is only about 50 ft-lb (68 Joules)--less than that of the .22 Short bullet. Energy is used efficiently by the sharp blade of the broadhead arrow. Cutting tissue is far more efficient than crushing it, and crushing it is far more efficient than tearing it apart by stretch (as in temporary cavitation).

5. Excision of the Wound as Not Only the Most Crucial but to Many the Sole Treatment for Gunshot Wounds:

"Debridement of missile injuries is essential to prevent clostridium myositis..." (7) is the often repeated military dogma. In many papers, administration of systemic antibiotics for the treatment of penetrating projectile wounds has been described as "only an ancillary

measure" (40), "an issue of debate" (41), or not mentioned at all (7). However, this dogma apparently overlooks the historical fact that the most important cause of death from missile wounds on the battlefield in the pre-antibiotic era was streptococcal bacteremia (70). Deaths from streptococcal bacteremia have been essentially eliminated from the battlefield by systemic antibiotics. A precipitous decline in the incidence of clostridium myositis, from 5% of those wounded in World War I to 0.7% in World War II and 0.08% in the Korean conflict (71), correlates very closely with the increasing use of antibiotics on the battlefield, yet debridement technique remained essentially unchanged during that time period. Thus, it appears that benefits of systemic antibiotic usage have been incorrectly attributed to wound debridement.

6. Spheres Assumed to Be a Valid Model for All Projectiles:

This misconception ignores the important variable of projectile shape. Comparing the wound profile produced by a sphere (Fig 4) with that produced by a military bullet (Fig 3) shows a basic difference in tissue disruption morphology. The maximum disruption produced by the sphere is always near the entrance hole, since projectile velocity is highest there. A pointed nondeforming bullet causes its maximum disruption not at the point of highest velocity, but where yaw increases the bullet's surface area striking the tissue (bullet shape becomes nonaerodynamic), causing increased tissue disruption. Although spheres may be useful in studying the effects of blunt fragments (like those from explosive devices), conclusions drawn from these studies are not valid when applied to bullet wounds.

7. Animals of 10 to 20 kg Falsely Assumed to Be a Valid Model for Human Wounds:

Temporary cavitation is no more than the pushing aside of tissue. The distance the tissue is displaced depends, among other things, on its weight. As might be expected, a given projectile will cause a temporary cavity of smaller diameter in a larger limb because of the increased weight of the mass being moved. This has been proved experimentally (72) and points out the misleading information that might be obtained through the use of these small animals. Bullet size cannot be reduced

without changing its characteristics, so there is no choice but to increase the size of the test animal to approximate the dimensions of adult humans if scientific validity is to be maintained.

8. Use of Tissue Simulants with Unproved Equivalence to Living Animal Tissue:

Fundamental to the use of tissue simulants, in lieu of animals, in wound ballistics is the establishment of their equivalence to animal tissue. For validity the simulant must reproduce the physical effects of the projectile-tissue interaction on the projectile (deformation, fragmentation), and in the simulant the projectile must stop at the same penetration depth as it does in living animal tissue. This requirement is frequently ignored by wound ballistics investigators (2, 28-30, 38) thus compromising, if not eliminating, the applicability of data so obtained to better understand the wounding process.

Duct-sealing compound (73), clay (2,74), soap (66, 72), gelatin (28-30, 38), and water-soaked phone books or newspapers (74) are commonly used tissue simulants. Information from each has been presented in the literature with the implication that it yields valid predictive information about wounding effects in living animals. Contrary to the assumptions that these materials are equivalent to animal tissue, bullet deformation caused by impact with them can vary widely. Recently, for example, we tested a 9-mm soft point pistol bullet that showed no deformation at all when shot into fresh swine cadaver leg muscle or into our 10% gelatin (shot at 4 degrees C), but expanded to a diameter of 15 mm when shot into duct-sealing compound (75).

Nonelastic tissue simulants (duct-sealing compound, clay, soap) can also mislead by their dramatic preservation of the maximum temporary cavity. Such demonstrations give a false impression that these cavities represent the potential for tissue destruction rather than the potential for tissue stretch. The latter may be absorbed by most living tissues with little or no lasting damage.

CONSEQUENCES OF THESE MISCONCEPTIONS

1. Inappropriate Treatment of Gunshot Wounds:

Sacrifice of viable tissue on the altar of "high velocity"--treatment more disruptive than the malady--is the most obvious consequence of the postulate that assumes that manifest tissue damage must accompany passage of a "high-velocity" missile. Surgical removal of excess tissue, based solely on a tenuous history of supposed projectile velocity, is practiced widely (34-40). In addition to the risk of permanent disability from excessive removal of muscle, such surgery takes longer with an attendant increase in surgical and anesthetic complications, and is more likely to require blood replacement.

In the battlefield setting the surgeon cannot know, with certainty, all the properties of the wounding projectile (shape, mass, construction type, striking velocity). In a majority of civilian cases information about the wounding weapon is not available (76). Fortunately, such information is not necessary for the proper treatment of gunshot wounds. In fact, it is the author's opinion that the patient will be better off if his medical care provider doesn't know anything about the wounding weapon at all. The provider might then, without bias, use objective data from his physical examination and roentgenographic studies to make more valid treatment decisions.

When a penetrating projectile does cause significant tissue disruption, that disruption is usually very obvious. For example, in an uncomplicated extremity wound caused by the M-16 rifle (Fig 2), if the bullet yaws significantly and fragments, this will be evident in the form of a large exit hole. If no significant yaw occurs, the exit will closely resemble the entrance hole, and little or no functional disturbance will be evident because of minimal tissue disruption. If, on the other hand, the bullet breaks up very early in its path through the tissue, it is possible that the entrance and exit holes could be small despite marked tissue disruption within the limb (such a pattern is typical of a soft point bullet (Fig 7); occasionally this pattern may also be produced by the M-16 bullet. The situation should pose no diagnostic problem; marked functional disturbance with swelling will be obvious on physical examination, and the

bullet fragmentation with soft tissue disruption will be obvious on biplanar x-rays. As in the therapy of any other form of trauma, objective data should guide treatment decisions.

The corollary postulate, "low-velocity projectiles cause insignificant damage," can also lead to disaster. The author was consulted recently about a case in which gas gangrene had developed in a leg wound caused by a .38 Special pistol (a "low-velocity" projectile). Surgical exploration of the wound had been delayed until 40 hours after the injury, and the first antibiotic had been administered four hours after the operation. It was the author's opinion that treatment had been inappropriate, but could not be considered negligent, since the literature contains many recommendations such as "...the majority of low velocity gunshot wounds of the extremities may be safely treated without recourse to the operating room" (41), and "Debridement is unnecessary for wounds caused by bullets whose muzzle energy is less than 400 foot pounds" (42). If antibiotic coverage had been started soon after the wound occurred, and if the bias obtained from the literature had not misled the surgeon to delay surgical exploration of the wound, this lethal infection most certainly would have been avoided.

2. Misguided Weapon Testing and Development:

A heavier bullet of lower initial velocity was recently adopted, by US military forces, to overcome deficiencies in the M-16 rifle's long-range performance. To stabilize this longer bullet the rifle's barrel had to be replaced by one with a faster rifling twist (causing the bullet to spin more rapidly). Not only was this change costly but it has produced a unique "error waiting to happen" situation. The new bullet is loaded in the same cartridge as the previous one. Thus it can be fired from the older M-16 rifles with the slower twist barrels. When this is done, the bullet is inadequately stabilized, resulting in extremely poor accuracy and yaw angles of up to 70 degrees in flight (77), possibly endangering the lives of soldiers who depend on it for protection on the field of battle. When fired from the new faster-twist barrel, it produces a wound profile similar to that of the older M-16 bullet, but when fired from the old barrel it causes marked tissue disruption at a shallower penetration depth (Fackler, M.L., unpublished data, 1984) much like a soft point bullet (Fig 8) (78).

Light bullets of high velocity lose velocity rapidly in flight--a basic physical phenomenon (11). Perhaps the aforementioned weapon problems could have been avoided if weapons designers had been less influenced by the mystique of "high velocity" and more influenced by basic physics of projectiles in flight. They might have realized that the older M-16 bullet was too light to be effective at longer ranges and used a heavier bullet in the first place. It is difficult to be optimistic for the future when these weapons developers still use the scientifically discredited "kinetic energy deposit" method to estimate wounding effects.

An extensive body of misinformation has been promulgated (28,29), based on the assumption that the temporary cavity produced by a handgun bullet is the sole factor determining its "incapacitation" effect on the human target. These studies were done to aid law enforcement agencies in their choice of weapons. The investigators superimposed temporary cavity measurements, derived from shots into gelatin blocks, on a "computer man" diagram of the human body. They judged relative damage by the anatomic regions "included" in the cavity. A "Relative Incapacitation Index" for each bullet was then calculated from these data. The superimposition of the temporary cavity on a region to determine the anatomic structures it encompasses reveals a serious misunderstanding of wounding mechanisms. By definition, no tissue is included "in" the temporary cavity; tissue is pushed aside by it. Using the permanent cavity in this fashion would make sense, but the permanent cavity is totally ignored in the calculation of the Relative Incapacitation Index. Not surprisingly, this Relative Incapacitation Index has been criticized (17, 79, 80), but reliance on its supposed validity continues to endanger the lives of those who must depend on the reliable performance of their weapon. These Relative Incapacitation Index studies were supported by the US Government (Dept. of Justice), causing many to assume their validity, and compounding the detrimental effects of the misinformation.

DISCUSSION

Violation of simple, fundamental scientific method appears to be the common thread that runs through the misconceptions dealt with in this review. The author has found verifiable validity in only a small percentage of the material in print. The field of wound ballistics is part physics and part biological science. Considering the large proportion of "exact" science in wound ballistics, we should expect to produce a literature with more validity and reproducibility than other medical or "inexact" fields. Quite the opposite appears to have taken place. Failure to consider all the variables in the missile-tissue interaction, failure to use a control animal, failure to calibrate tissue simulants, failure to require data to support assumptions, etc.--these were the basic errors responsible for the misconceptions listed in the foregoing pages. The reader will probably agree that none of them involve a high degree of complexity.

Misinterpretation of war trauma experience has misled many writers. Such experience is anecdotal. Rarely if ever is the weapon, type of bullet, distance from muzzle to target, and absence of intermediate targets known with certainty on the battlefield as it is in the wound ballistics laboratory. Memory mixes all types of war wounds together, assumptions on treatment efficacy are made despite lack of follow-up information, and statements from higher headquarters concerning treatment rendered in the field of action are frequently based on inaccurate data and incorrect assumptions. In sum, a lot of error is reported as fact.

Physicians writing in the field of wound ballistics need to acquire sufficient expertise in weapon technology so that they are not completely dependent on ballistics engineers or other "experts" for information. Ballistics engineers writing in the field must acquire sufficient expertise about the living animal so that they at least know the pertinent questions to ask. Unless the "knowledge gap" between the physical and biological sciences is bridged at least partially by those who work in this field, an enormous potential for inaccuracy is likely to continue.

Recognizing the projectile-tissue interaction as a simple mechanical collision and comprehending how tissue is disrupted (crush and stretch) in this collision,

coupled with wound profiles illustrating how much crush and stretch occurs at any depth of projectile penetration, should give the reader sufficient background to recognize any perpetuation of past errors or creation of new ones in the future. It is not surprising that attempts to teach wound ballistics using formulae or tables of velocity and kinetic energy have been counterproductive. These methods have diverted attention from the actual tissue disruption and made the subject appear unnecessarily complicated.

An intelligent surgeon, knowing nothing about gunshot wounds except that they are contaminated, would most likely treat them quite appropriately. He would base his treatment decisions on objective data from the physical examination and x-ray studies, as he would in treating any other form of trauma. The surgeon who has read and accepted what is written in the wound ballistics literature could become a menace, doing more harm with his treatment than was done by the bullet. It is encouraging to note from the author's own experience as a combat surgeon and contacts with others that most treatment of penetrating injuries rendered on the field of battle was governed more by the common sense and good training of the surgeon than by what is written in the wound ballistics literature.

ACKNOWLEDGEMENTS The author wishes tc acknowledge the advice and assistance of John D. O'Benar, PhD, and Charles E. Wade, PhD, of the Military Trauma Research Division, and John P. Hannon, PhD, Scientific Advisor of the Letterman Army Institute of Research, in arranging the data and expressing the thoughts contained in this paper. He also wishes to express appreciation to Paul J. Dougherty, Senior Medical Student at the Uniformed Services University of the Health Sciences Medical School, for his contribution of valuable literature references previously unknown to the author.

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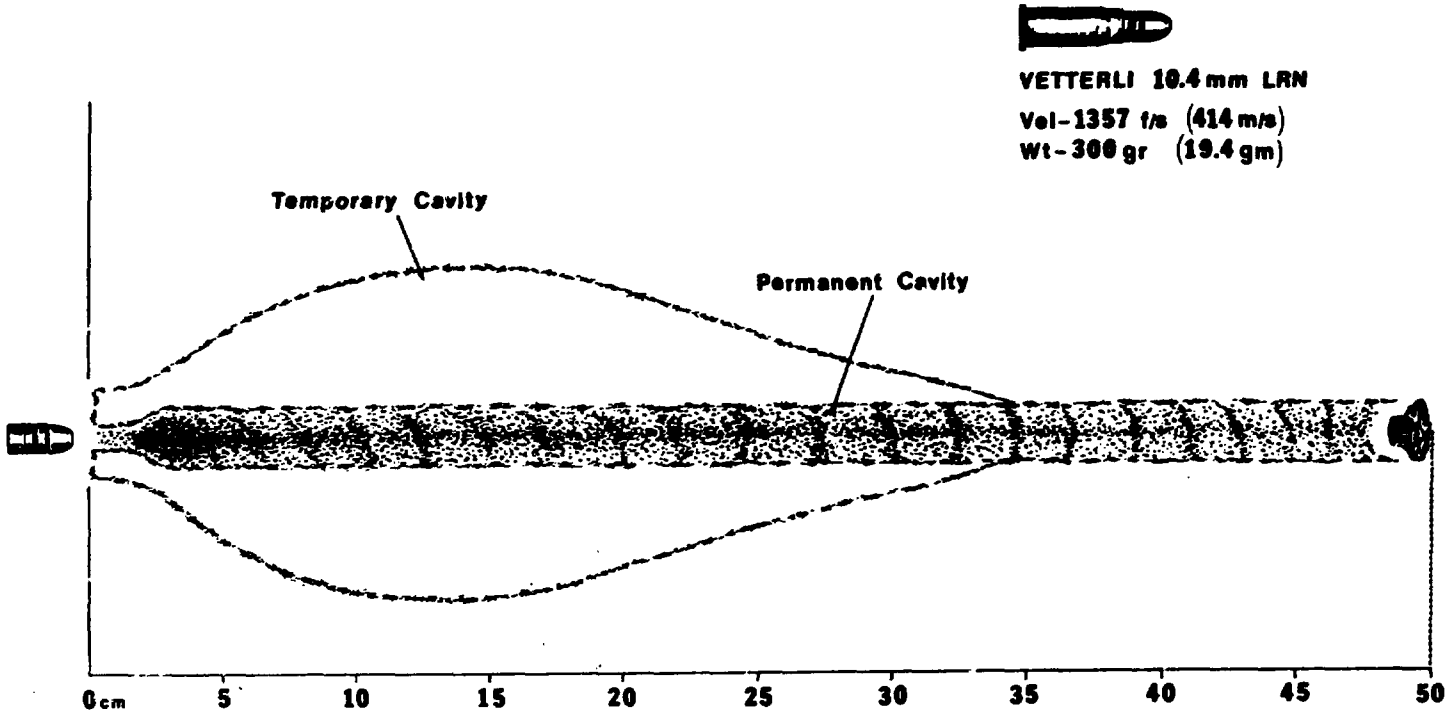

Fig 1. --The large lead Vetterli rifle bullet is typical of military bullets circa 1850-1890. Bullet deformation upon striking tissue caused a large permanent cavity. A sizable temporary cavity was also produced, despite a velocity less than half that of modern military rifles.

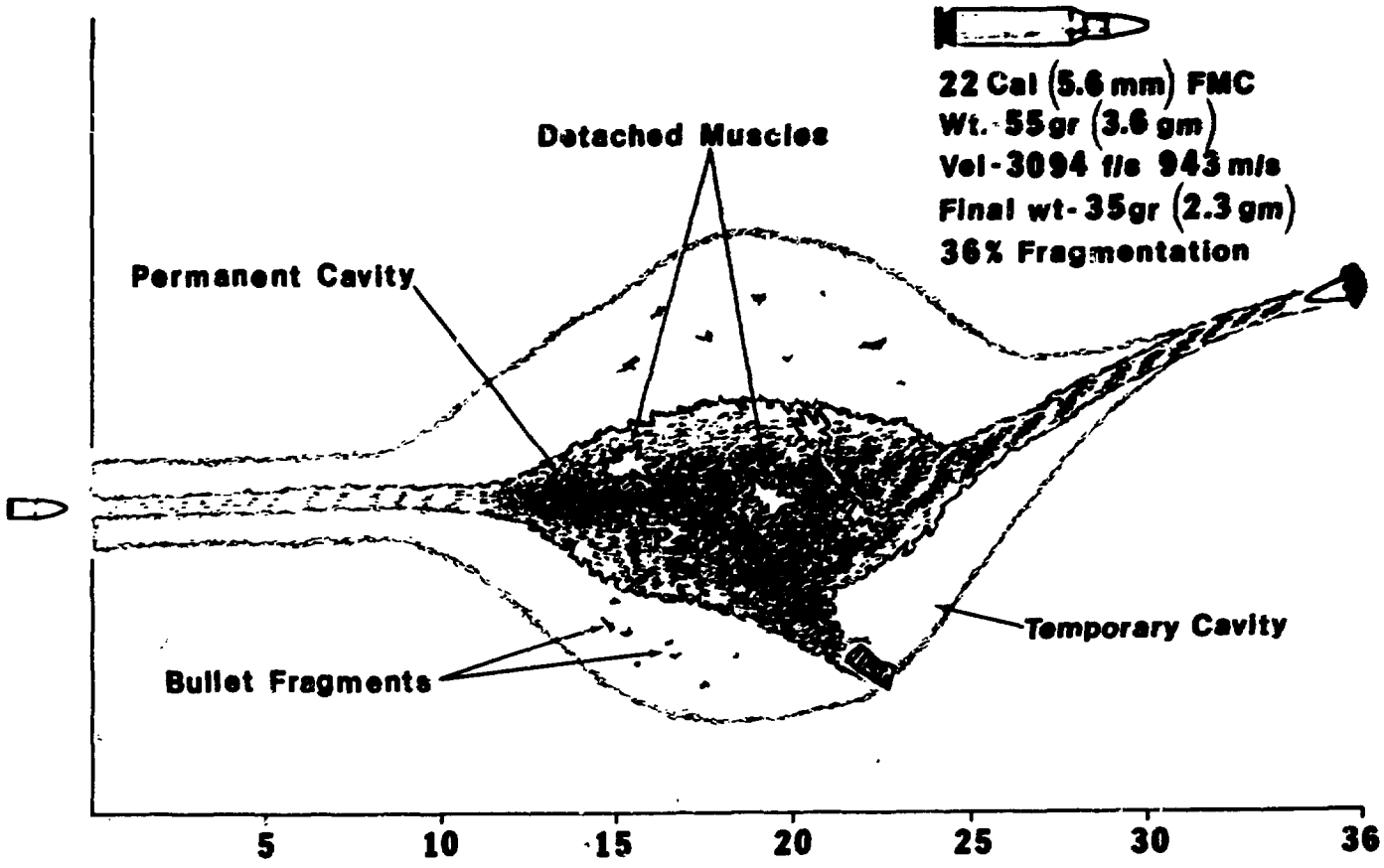

Fig 2. --The marked tissue disruption produced by the M-16 bullet occurs most often at a penetration depth of 15 to 25 cm. The minimal disruption produced in the first part of the tissue path explains the confusion surrounding this weapon's effects.

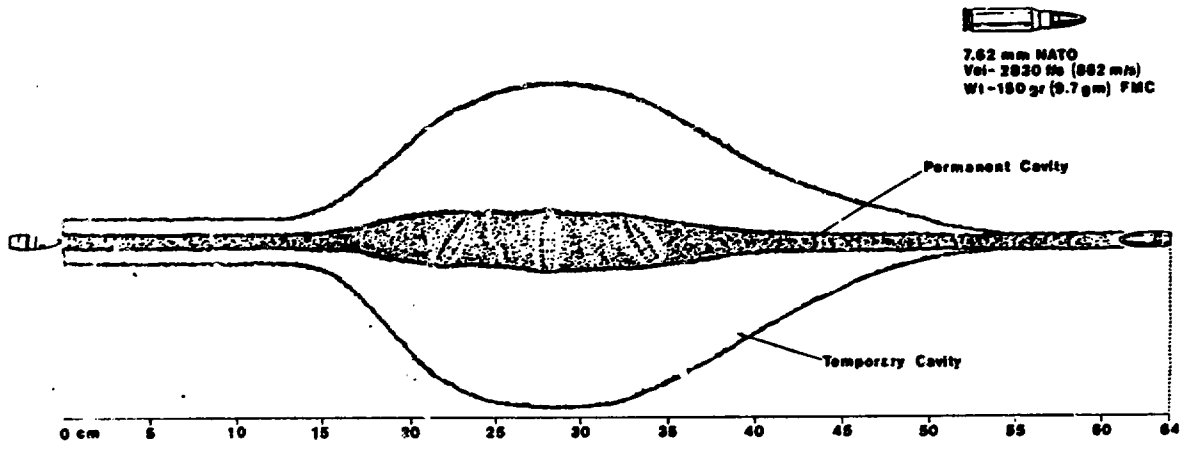

Fig 3. --The military 7.62 NATO bullet yaws after 15 cm of penetration but remains undeformed. Maximal tissue disruption occurs at the point of 90 degree bullet yaw (penetration depth circa 28 cm) rather than at the point of highest bullet velocity (tissue surface).

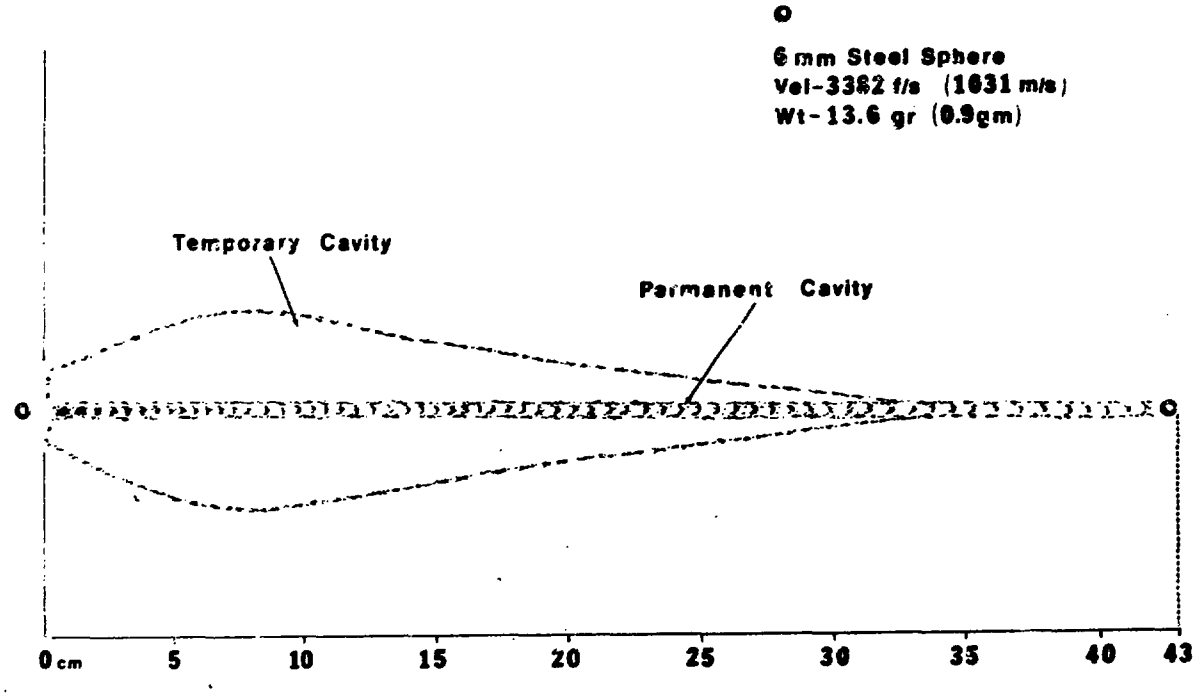

Fig 4. --The 6-mm steel sphere causes its maximum disruption in the first 10 cm of penetration. Since the surface of a sphere striking tissue cannot increase by yaw, temporary cavity size corresponds to projectile velocity.

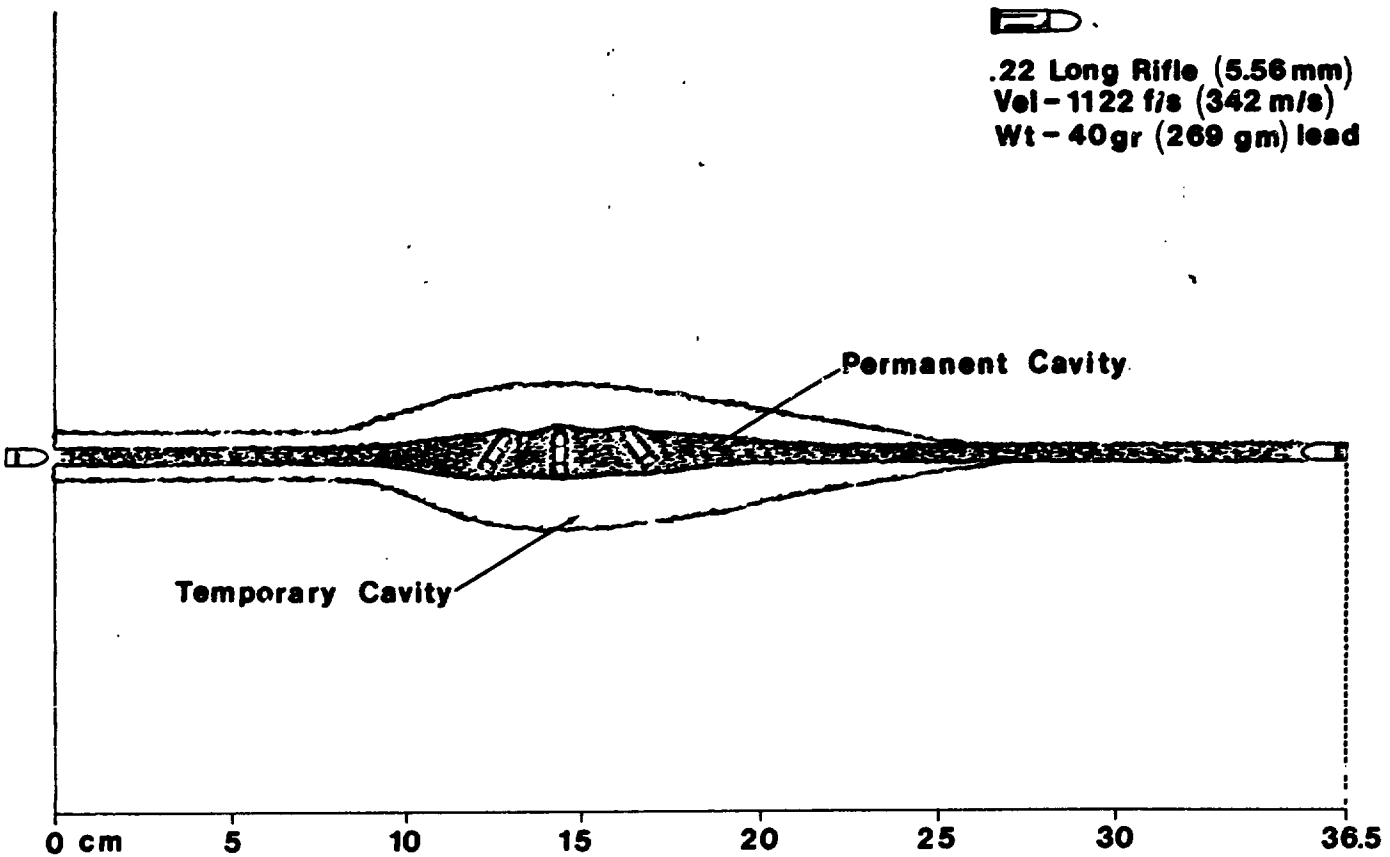

Fig 5. --The .22 Long Rifle bullet does not deform upon striking soft tissue. Significant yaw occurs after a penetration depth of circa 8 cm, and the bullet ends up traveling base forward for the last half of its tissue penetration.

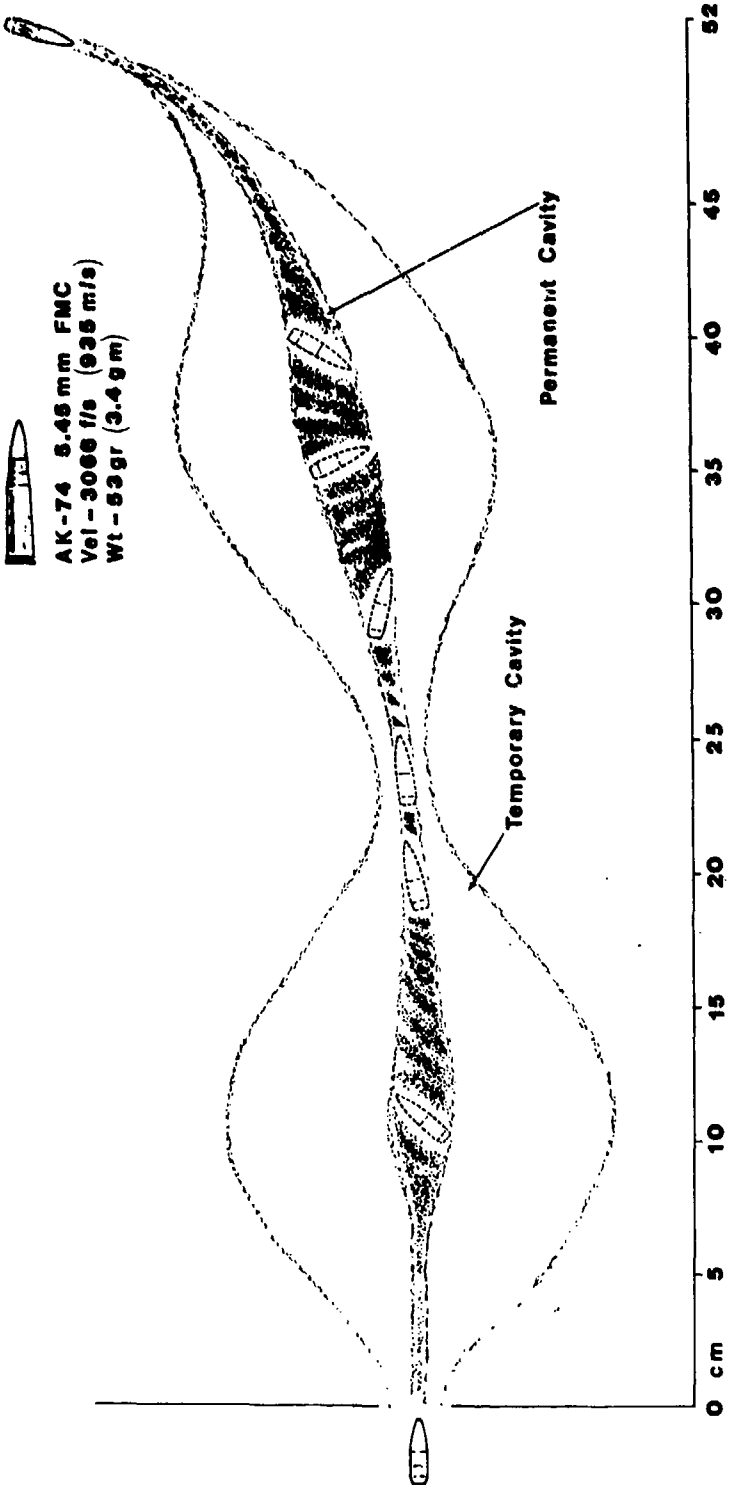

Fig 6. --The AK-74 is the Russian contribution to the new generation of military assault rifles. The bullet typically yaws at a shallower penetration depth than other military bullets. This results in a significant wound even in uncomplicated extremity hits.

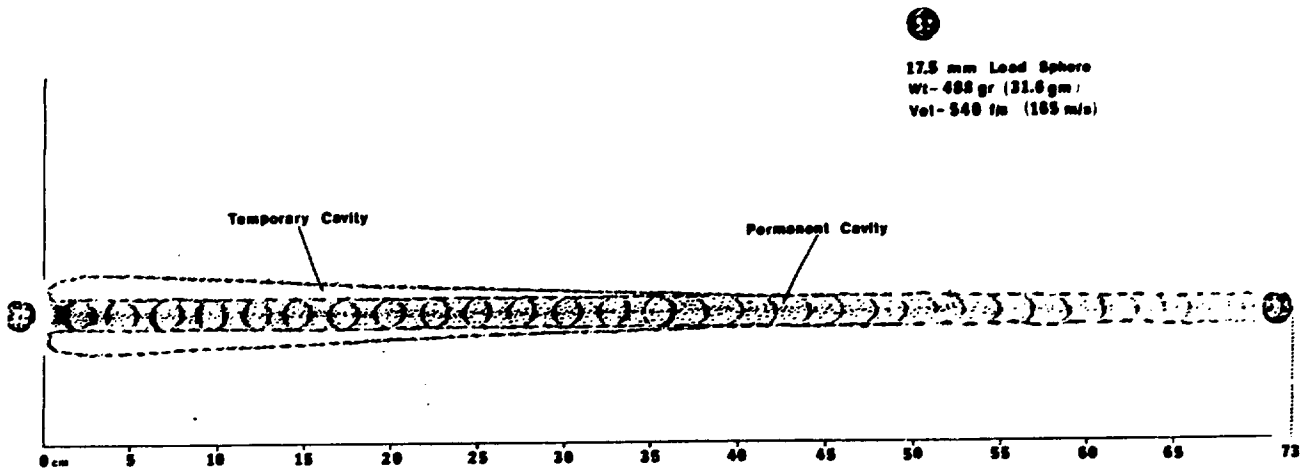

Fig 7. --The 17.5-mm lead sphere, striking at 540 ft/s (165 m/s) duplicates the kinetic energy of the 6-mm steel sphere (Fig 4). The large sphere penetrates 30 cm deeper, and produces a permanent cavity over 50 times the volume of that produced by the smaller sphere.

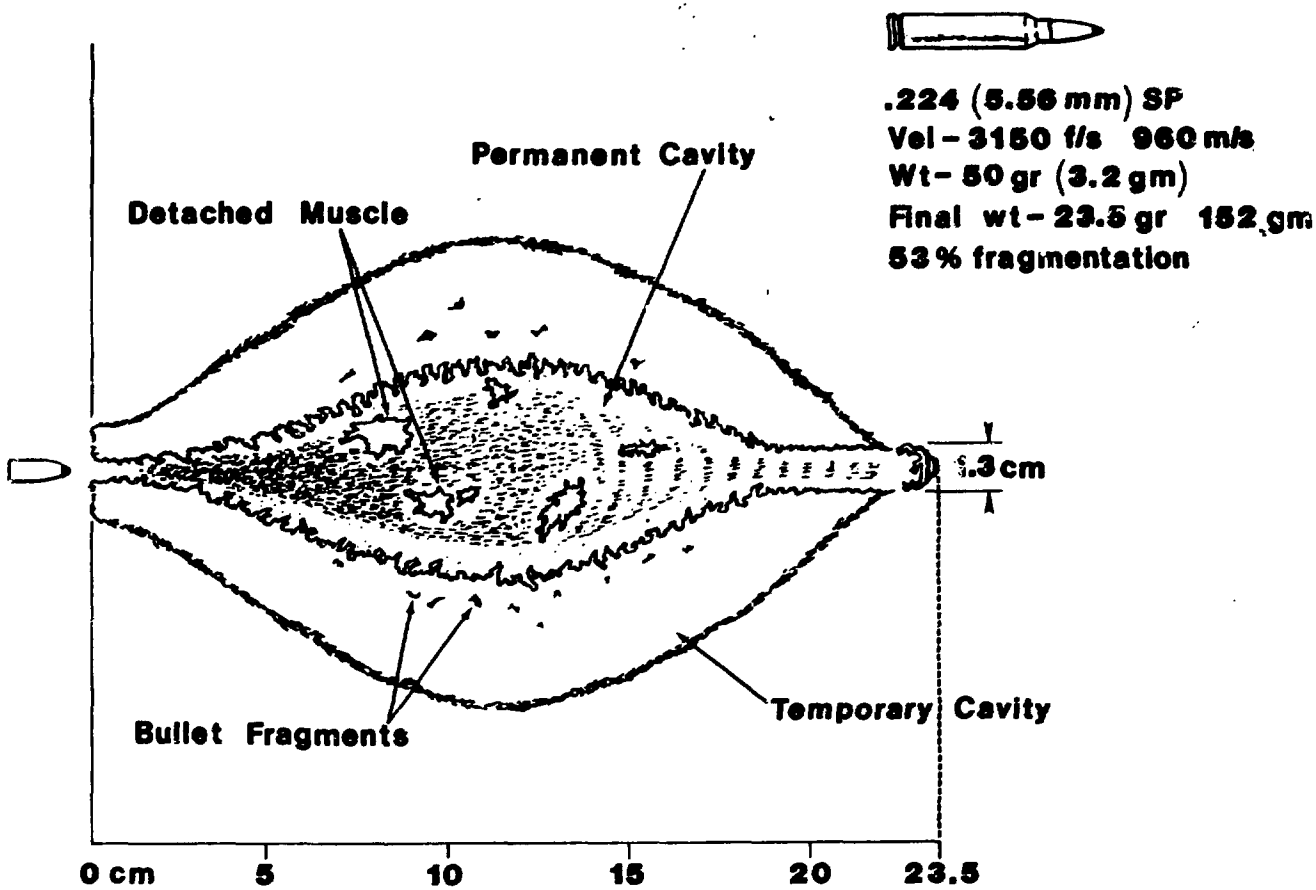

Fig 8. --The hollow-point construction of this 22 caliber bullet causes it to break up during the first 10 cm of tissue penetration. The tissue weakening by multiple bullet fragments interacts with temporary cavity stretch to cause increased permanent tissue disruption.

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